A NEUROPATHOLOGICAL STUDY OF THE MYENTERIC PLEXUS IN GASTRIC PTOSIS

AUTHOR(S):

YAMAUCHI, AKIRA

CITATION:

YAMAUCHI, AKIRA. A NEUROPATHOLOGICAL STUDY OF THE MYENTERIC PLEXUS IN GASTRIC PTOSIS. 日本外科宝函 1958, 27(6): 1325-1332

ISSUE DATE:

1958-11-01

URL:

http://hdl.handle.net/2433/206721

A NEUROPATHOLOGICAL STUDY OF THE MYENTERIC PLEXUS IN GASTRIC PTOSIS

by

AKIRA YAMAUCHI

Second Surgical Division, Kyoto University Medical School
(Director: Prof. Dr. YASUMASA AOYAGI)
Received for publication Apr. 22, 1958

I. INTRODUCTION

The neuropathological changes in the stomach in various diseases were reported by Ph. STÖHR Jr., W. RIEDER, V. BERGMANN, PERMANN, YAMADA and others. YAMADA studied the morphological changes of the nerve cells and the nerve fibers in the stomach in gastric ulcer, cancer of the stomach and chronic gastritis.

The author has studied in this study the changes in the nervous elements in the stomach of patients with gastric ptosis.

Gastric ptosis sometimes accompanies chronic gastritis or gastric ulcer, or it may occur as a part of general splanchnic ptosis.

YAMADA of our clinic reported a hyperplastic and degenerative change in the myelinated gastric nerves (he described as sensory in nature) and various degenerative figures in the autonomic nerve elements in AUERBACH's plexus and proliferation of peripheral networks at the bottom of gastric ulcer and chronic gastritis. The author, therefore, studied the other form of gastric ptosis which occurs as a part of general splanchnic ptosis.

II. MATERIALS AND METHODS

Fresh stomachs, resected for ptosis, were immediately fixed in 10% neutral formol solution for about 2 weeks, sliced into 35-45μ by the freezing method and were kept in the formol solution for 2 or 3 more weeks, and then they were impregnated.

BIELSCHOWSKY's silver carbonate method modified by Jabonero was used.

III. MICROSCOPIC OBSERVATIONS

The morphological change was investigated mainly in the nerve elements of AUERBACH's plexus. In the ptotic stomach, the nervous apparatus presented a slight degree of regressive degeneration. This change was found only in a part of the gastric wall where there was most marked dilatation, being absent in the pylorus or other non-dilated portions.

1. Changes in the nerve cells

In the distended corpus of the stomach, a slight decrease in the number of DOGIEL's type I nerve cells was observed. The nerve cells showed varying degrees of degeneration such as dislocation of the nucleus and changes of the neuroplasm.

a) Changes in the nucleus

Marginal dislocation of the nucleus (called "Kernrandstellung" by FEYRTER) was found in many nerve cells; the nuclei dislocated to a marginal zone of the nerve cells sometimes looking as if the nuclei escaped from the cell body. Only a very few nuclei were destroyed.

Deformed nuclei and changes in nucleoli were not seen.

b) Changes in the protoplasm of the nerve cell

The main change was a rough structure and granulated degeneration of the protoplasm. Besides these, a hyperchromatic change with or without shrunken protoplasm was seen frequently.

Vacuoles in the protoplasm of the nerve cells with windows (Fensterzelle) were observed, though seldom.

c) The dysharmonious nerve process (Fortsatz dysharmonie) of STÖHR was observed in the nerve cells: nerve cells with many fine nerve processes which grew out irregularly from cell bodies, or with bundles of nerve processes arising from a part of a nerve cell.

2. There was less change in the nerve fibers than in the nerve cell.

The nerve bundles in AUERBACH'S plexus did not present any change, but the peripheral networks which were distributed directly to the muscle fibers (Auerbach's plexus order Ⅲ.) had disappeared.

The so called corkscrew feature of a degenerated fiber was found in the muscular layer of the gastric corpus.

3. Changes in accessory cells.

Changes in the accessory cells were not seen except for a slight increase in number around some of the degenerated nerve cells.

In Meissner's plexus, no change in the nerve cells was seen. Only a few nerve fibers showed spindle-shaped irregular swellings along their courses.

DISCUSSION

The pathological changes in the nervous elements found in ptotic stomachs accompanying general splanchnic ptosis were always regressive ones.

These changes are clearly different from those seen in ptotic stomachs with chronic gastritis or gastric ulcer. The latter presents, as Yamada described, hyperplastic features of the nerve elements such as hyperplastic changes in the nerve fibers and proliferation of the autonomic nervous networks besides regressive processes.

In other words, chronic inflammation of the stomach first causes irritation of the gastric nerves resulting in gastric pain or spastic changes in the muscle tone of the stomach, while the secondary changes, which are presented as regressive processes, cause a lowered tone of the gastric muscles as well as weak peristalsis.

Y. WATANABE of our clinic studied the visceral reflexes of the alimentary canal. He applied acetylcholin solution to the wall of the stomach or the intestine, which caused spasm of the stimulated area and lowered tone in the other portions

of the alimentary canal. He came to the conclusion that stimulation of a part of the alimentary canal, though it may evoke a spasm in the stimulated part, can cause a general ptotic appearance of the alimentary canal. According to his view, the stimulated features of the nerve elements in the stomach with chronic inflammation may be able to cause apparent ptotic stomach. On the other hand the stimulated myenteric plexus may cause irregular peristalsis resulting in gastric spasm. KIMURA maintains that the ptotic stomach with colicky pain suggests gastric ulcer or a preulcerous change in the stomach and he differentiated these from ptotic stomachs with general splanchnic ptosis.

The author's results, in accordance with YAMADA's neurohistological study on gastric inflammations, agree with the different nature of these two forms of gastric ptosis.

The neuropathologic change in gastric ptosis accompanied by general splanchnic ptosis is much less than gastric ptosis with chronic gastric inflammation. The changes in the former fail to show any stimulation of the gastric nerve elements. The author could find many normal nerve cells besides degenerated ones scattered among them.

DOGIEL divided the nerve cells in the AUERBACH's plexus into two groups; types I and II. Considering the physiological significance of these nerve cells, Feyrter and JABONERO believed Dogiel's type I to be motor and type II sensory in nature. Honjin et al. divided the nerve cells into two groups by their affinity to silver, i. e. one with deeply stained cells and one with weakly stained cells.

R. Inoue, in our clinic, has the following opinion on the degenerating process of the nerve cells. He supposes that DOGIEL's type I nerve cells present, in the process of degeneration, hyperstainablility, changes in number and form of the nerve process and shrunken cell bodies which, gradually assuming a granular structure, finally are destroyed and disappear. The nuclei in these cells are dislocated first to a margin of the cell body and sometimes fall out of the cell body. DOGIEL's type II nerve cells, which have poor stainability in the normal state, present a swelling of the cell body without showing any tendency to shrink or hyperstainability. In the nuclei of DOGIEL's type II nerve cells neither dislocation nor escape from the cell body are observable in the degenerating process.

In general, according to INOUE, DOGIEL's type II nerve cell is much more resistant to pathological stimulation than type I.

The author found degenration only in DOGIEL's type I nerve cells.

This suggests that neuropathologic change is not severe. The degeneration of DOGIEL's type I nerve cells and the poor stainability in the fibrils of the tertiary AUERBACH'S plexus resemble changes in the nerve elements found in the mobile cecum by R. INOUE.

This may be due to the stagnation of the contents in the ptotic stomach. The same change was reported by R. INOUE in the colon of patients suffering from chronic constipation or descending megacolon.

From the common features of neuropathological changes in these disease, the

author concludes that the ptotic stomach accompanied by general splanchnic ptosis presents pathologic symptoms from the functional disturbance of the intrinsic nerve elements.

NAKAYAMA (University of Chiba), from the digestive and resorptive function of the stomach, maintains that in the ptotic stomach gastric resection may be done only on the gastric corpus. From the neuropathologic point of view, the author agrees with his opinion. The pyloric portion which has no pathologic change can be left unremoved in gastric resection.

V. SUMMARY AND CONCLUSIONS

From the neurohistological study of the ptotic stomach accompanied by general splanchnic ptosis, the author presents the following conclusions.

(1) A slight pathological change was found in AUERBACH's plexus.

(2) The pathological change was observed only in the dilated portion of the gastric corpus, and none was found elsewhere.

(3) The nerve cells in which the pathological change was observed, were restricted to DOGIEL's type I nerve cell.

(4) The networks in the III AUERBACH's plexus showed poor stainability or had disappeared.

(5) This disease is believed to be dysfunction of the intrinsic nerve elements which are considered parasympathetic in origin.

References

1) Clara, M.: Das Nervensystem des Menschen. Leipzig. I. A. Barth. 1942. 2) Feyrter, F.: Ueber die Pathologie der Vegetativen Nervösen Peripherie und ihrer Ganglionören Regulationssatätten. 1951. 3) Herzog, E.: Acta Neuro-Veg. **10**, 110, 1954. 4) Jabonero, V.: Der Anatomischen Aufbau des Peripheren Neuro-vegetativen Systems. Acta Neuro-Veg. Suppl. 4, 1953. 5) Kimura, Ch.: J. Jap. Surg. Soc. **57**, 6, 1956. 6) Lee, I, M.: Arch. f. Jap. Chir. **25**, 3, 1956. 7) Miyake, H.: Tokyo med. J. 2998, 1936. 8) Nakamura, T. (Nakayama clinic): Surgery **18**, 7, 1956. 9) Osaki, T.: Fukushima J. Med. Science. **4**, 3-4, 1954. 10) Otsu, A.: Acta Sch. Med. Univ. Kyoto, Jap. 31, 1953. 11) Reiser, K. A.: Z. Bl. f. Zellforsh. **15**, 1761, 1932. 12) Reiser, K. A.: Mikroskopische Untersuchungen des intramularen Plexus bei chirugischen Erkrankungen des Magens. Deutsche Z. Chir. **244**. 471, 1953. 13) Sekigawa, J.: Juzenkai Zasshi. (in Japaness) **41**, 2442, 1936. 14) Stoehr, Jr, P.: Studien zur normalen und pathologischen Histologie vegetativen Ganglien. I und II. Z. Zellf. 32, 587, 1943. **33**, 109, 1944. 15) Stoehr, Jr, P.: Acta Neuro-Veg. **10**, 21, 1954. 16) Yamada, H.: Arch. f. jap. Chir. **26**, 2, 1957. 17) Watanabe, Y.: Arch. f. jap. Chir. **23**, 1954.

和 文 抄 録

胃下垂症に於ける筋間神経叢の神経病理学的研究

京都大学医学部外科学教室第2講座（青柳安誠教授 指導）

山 内 晧

JABONERO 氏鍍銀染色法を用いて，一般内臓下垂症の際の下垂胃に対する胃切除標本について筋間神経叢の態度を追求し次の如き結果を得た．

1) ア氏神経叢に軽度の変化が見られた

2) 変化は胃体部にのみ見られ，他の胃部には見られなかつた

3) 変性の見られた神経細胞は DOGIEL I 型に属する神経細胞であつた

4) 第3次ア氏神経叢たる筋線維支配の終網は殆ど見られないか，或は全く消失していた

5) 本症は副交感性神経要素の失調と考へられる．

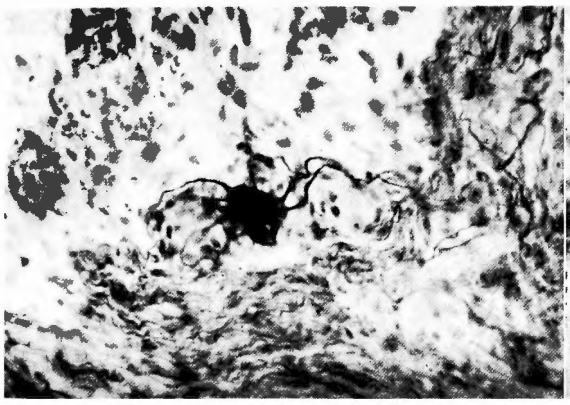

Fig. 1. The DOGIEL'S II type nerve cell. ×400

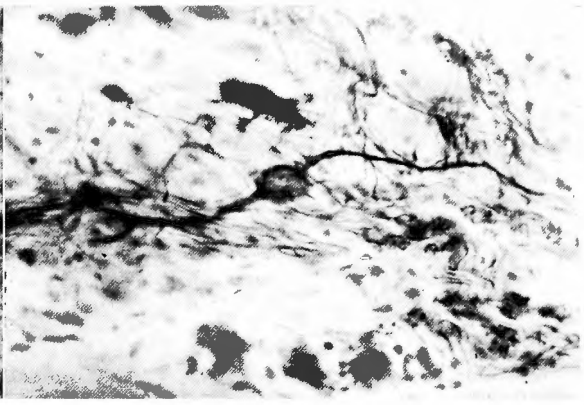

Fig. 2. The FEYRTER'S III type nerve cell. Two long nerve process are observed. ×400

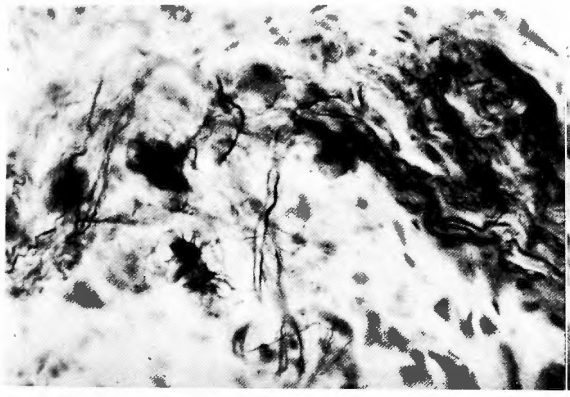

Fig. 3. Degenerated nerve cells in the AUERBACH'S plexus. Dislocated nuclei are observed. ×400

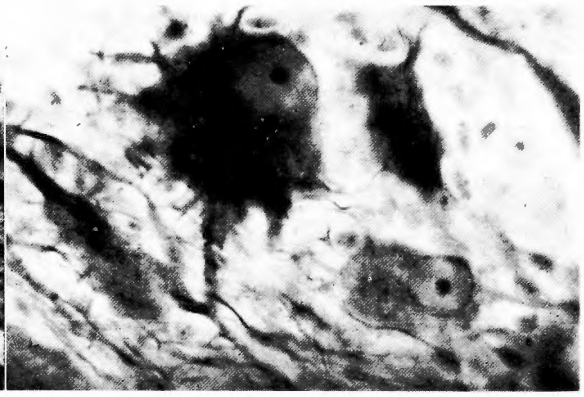

Fig. 4. Dislocation of the nucleus to a marginal zone of a nevre cell. ×1000

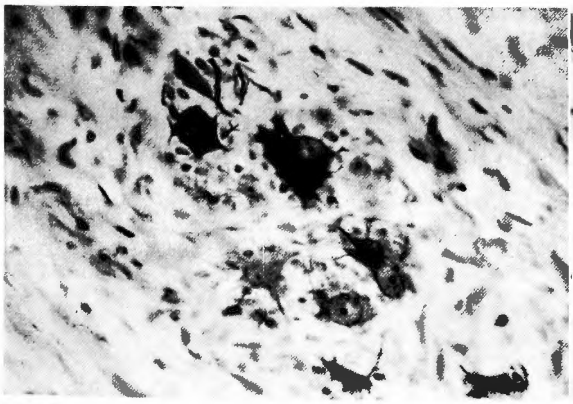

Fig. 5. A group of degenerated nerve cells in the AUERBACH'S plexus. ×400

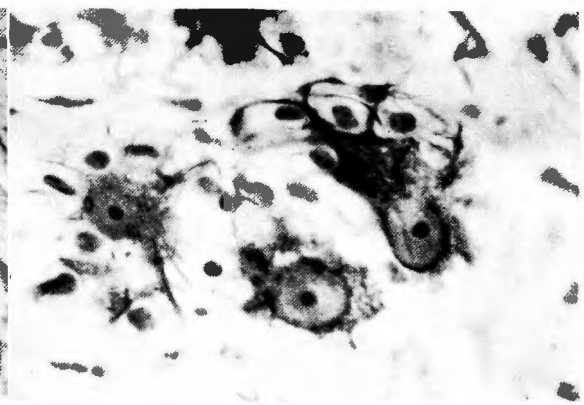

Fig. 6. Enlarged photo of fig. 5. Dislocation of nuclei to the marginal zone of the nerve cells. ×1000

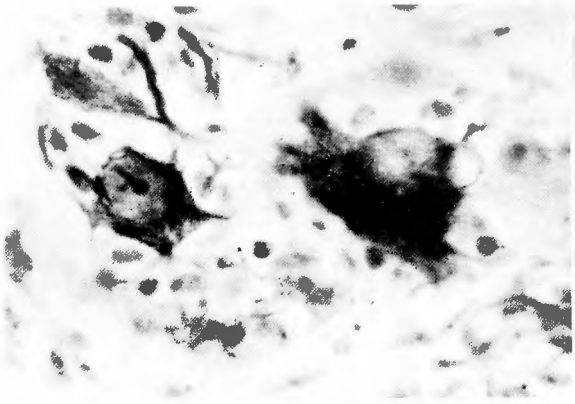

Fig. 7. Enlarged photo of fig. 5. Hyperchromasy and shrinkage of the protoplasm in the nerve cells. ×1000

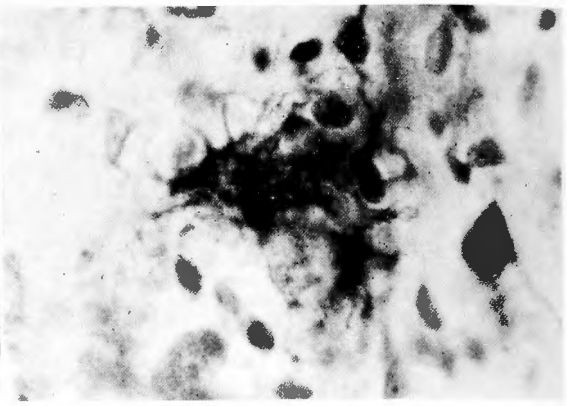

Fig. 8. A rough structure of a nerve cell in the AUERBACH'S plexus. ×1000

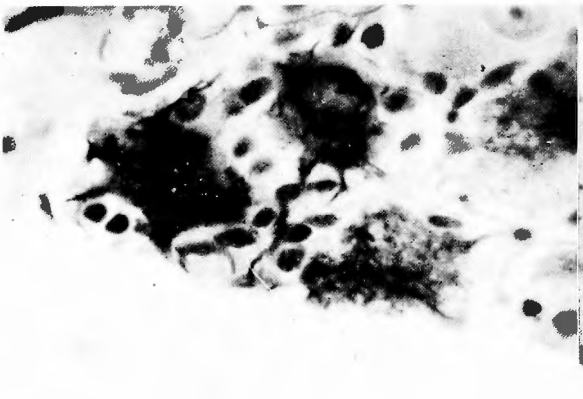

Fig. 9. Degenerated nerve cells in the Auerbach's plexus. Deeply stained protoplasm shows a rough structure. ×1000

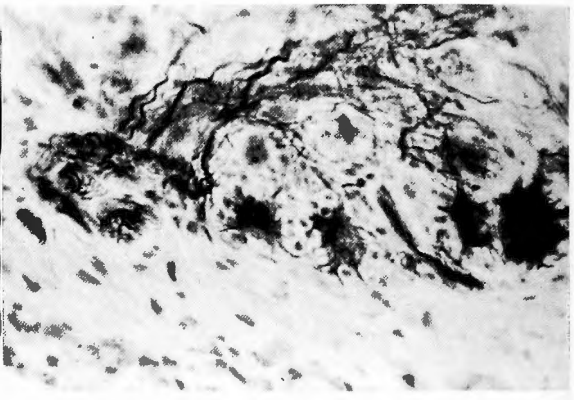

Fig. 10. Normal nerve fibers running around the degenerated nerve cells in the AUERBACH'S plexus. ×400

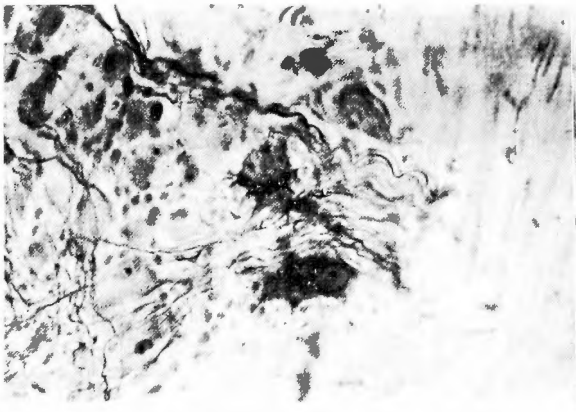

Fig. 11. Degenerated nerve cells showing the dislocation of nuclei to a marginal zone of nerve cell body. The protoplasm is deeply stained. ×400

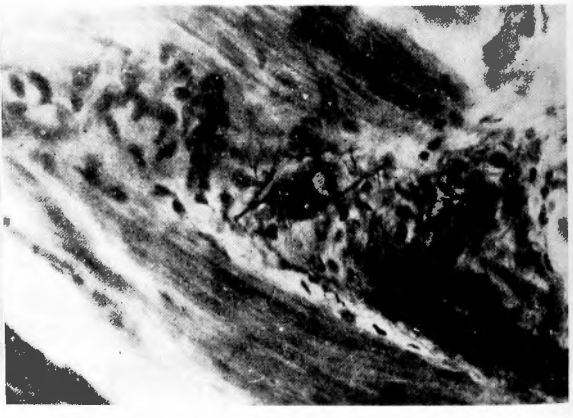

Fig. 12. A nerve cell with a window in the AUERBACH'S plexus. ×400

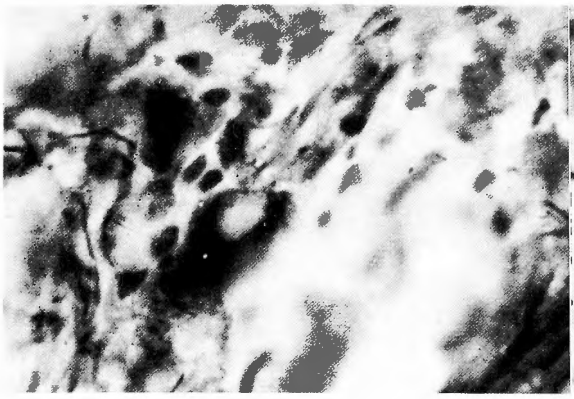

Fig. 13. A nerve cell with a large vacuole in the protoplasm. AUERBACH'S plexus. ×1000

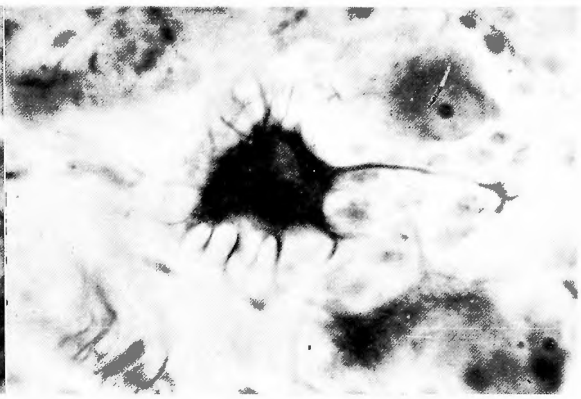

Fig. 14. A degenerated nerve cell showing disharmonious growth of the nerve process. ×1000

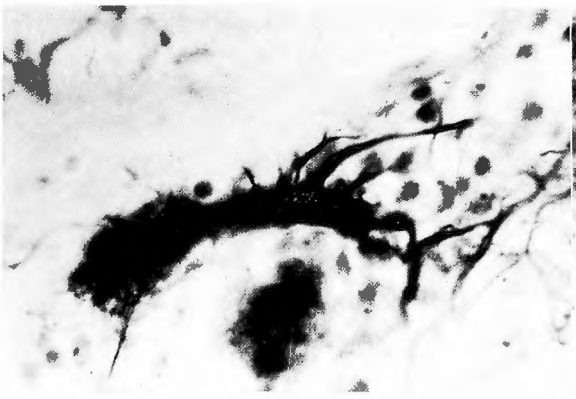

Fig. 15. A disharmonious growth of the nerve process from a degenerated nerve cell in the AUERBACH'S plexus. ×1000

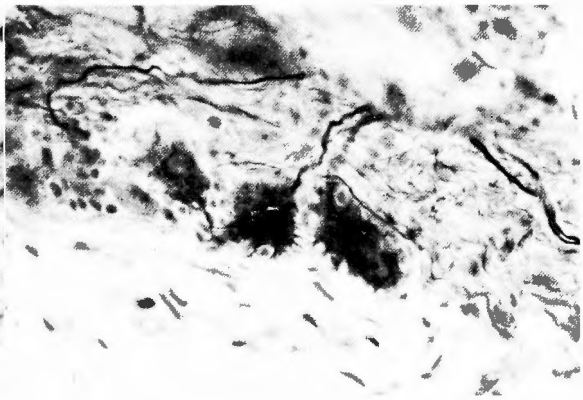

Fig. 16. Degenerated nerve cells and the normal nerve fibers running around them. ×1000

Fig. 17. A corkscrew-like nerve fiber in the muscular layer. ×400

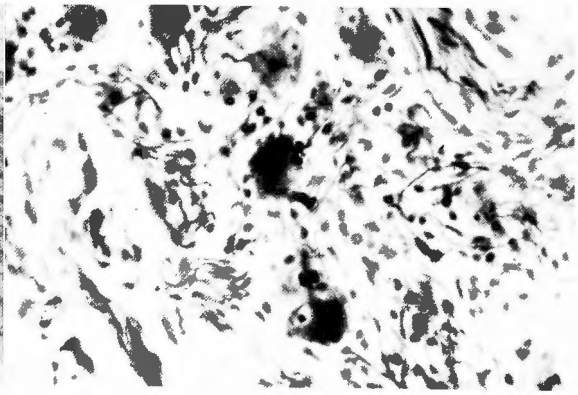

Fig. 18. Degenerated nerve cells showing the marginal dislocation of nuclei and the normal nerve fibers around them. ×400

Fig. 19. A degenerated nerve fiber with irregular swelling in the MEISNER'S plexus. ×400